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Impact of Obesity on Venous Hemodynamics of the Lower Limbs

INAUGURAL-DISSERTATION

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1. Abstract

Background: Obesity is a risk factor for chronic venous insufficiency and venous thromboembolism. Aim of this study was to compare venous flow parameters of the lower limbs assessed by duplex scan in obese and non-obese subjects.

Methods: Venous hemodynamic was studied in non-obese (BMI <25 kg/m²) and obese (BMI>30 kg/m²) subjects by color-coded duplex sonography. Diameter, flow volume, peak, mean and minimum velocities were assessed in the femoral vein.

Results: 46 non-obese limbs (23 subjects) and 44 obese limbs (22 subjects) were examined. Diameter of the femoral vein was significantly greater in obese compared to non-obese subjects (8.5 ± 2.2 mm versus 7.1 ± 1.6 mm, $p= 0.0009$). Venous peak and minimum velocities differed between non-obese and obese subjects (14.8 ± 7.2 cm/s vs. 10.8 ± 4.8 cm/s, $p=0.0071$ and -4.0 ± 3.6 cm/s versus 1.7 ± 6.3 cm/s, $p=0.056$). Calculation of venous amplitude and shear stress showed significantly higher values in non-obese compared to obese subjects (18.8 ± 9.4 cm/s versus 12.5 ± 9.3 cm/s, $p=0.003$ and 2.13 ± 2.2 dyn/cm² versus 1.6 ± 2.7 dyn/cm², $p=0.03$). Spearman rank correlation revealed a significant inverse correlation between waist-to-hip ratio as well as the waist circumference and venous peak velocity, mean velocity, velocity amplitude (peak velocity – minimum velocity) and shear stress.

Conclusion: Lower limb venous flow parameters differ significantly between healthy obese and non-obese subjects. These findings support the mechanical role of

abdominal adipose tissue potentially leading to elevated risk for both venous thromboembolism and chronic venous insufficiency.

2. Introduction

Several epidemiological studies have given strong advice to the hypothesis that obesity is a risk factor for both chronic venous insufficiency (CVI) and venous thromboembolism (VTE).¹⁻³ Obesity plays a key role in the development of metabolic syndrome with a cluster of cardiovascular risk factors.⁴ Excess body weight has also been related to various alterations in the coagulation system including impaired fibrinolytic activity and elevated plasma concentrations of clotting factors.⁵ These alterations in endothelial function and coagulation are thought not only to be relevant for arterial but also for venous thrombosis.⁶ In addition to these mechanisms, obesity is thought to predispose venous stasis, which is a trigger of both deep VTE and CVI. Central abdominal obesity is thought to be associated with an increased intraabdominal pressure (IAP) caused by abdominal fat.⁷⁻⁹ Arvidsson and coworkers showed that the pressure in the iliofemoral vein in morbidly obese patients is significantly higher compared to non-obese subjects.⁸ Likewise, it has been shown that surgical weight reduction decreases urinary bladder pressure, a surrogate marker of IAP.⁹

Elevated IAP therefore might impede venous backflow in the iliofemoral veins resulting in venous stasis and vein distension of the lower limbs favouring valve dysfunction and thrombosis. To our knowledge there are no human in-vivo studies aimed at proving this assumption. Color-coded duplex sonography (CCDS) allows a non-invasive and accurate flow assessment of the veins of the lower limbs.¹⁰ We hypothesized that venous flow characteristics of the lower limbs differ between obese and non-obese subjects inasmuch as obese subjects exhibit lower flow velocities and larger vein diameter and that abdominal obesity correlates with venous hemodynamic changes.

3. Methods

Subjects and Study Protocol

Venous hemodynamic of the lower limbs was studied in 46 limbs of non-obese (BMI < 25 kg/m²) and 44 limbs of obese (BMI >30 kg/m²) otherwise healthy subjects.

Inclusion criteria were either BMI < 25 kg/m² or >30 kg/m². Exclusion criteria were clinical signs of CVI, duplex sonographic evidence of venous valve dysfunction or outflow obstruction, prior limb surgery or sclerotherapy, history or clinical signs of arterial disease, connective tissue disorders, leg trauma or swelling, clinical signs of lymphedema, history of chronic obstructive pulmonary disease and cardiac failure.

The protocol entailed patient history, clinical examination and duplex sonography to exclude venous incompetence or thrombosis. The local ethic committee of the University Hospital of Zurich approved the study (EK 1709) and all patients gave written informed consent. Medical staff and students volunteered as study participants.

Body Mass Index and Waist-to-Hip Ratio

BMI was calculated from measured patients height and weight using the following equation: BMI = kg/m².

Waist circumference was measured at a point midway between the costal margin and iliac crest and in line with mid-axilla. To measure hip circumference the greater trochanter was located as the widest part of the hips at the level of buttock line. Both waist and hip circumference were assessed in upright position. Afterwards the waist-to-hip-ratio (WHR) was calculated.

Color-coded duplex sonography (CCDS)

Duplex assessments were performed in the vascular laboratory of a tertiary referral hospital by experienced vascular physicians. Standardized supine position was applied for all duplex scan measurements with subjects in upper body elevation of 10^0 .

CCDS was performed by using the Accuson Sequoia 512 (Siemens AG, Medical Solutions, Zurich, Switzerland) with a 8 MHz linear scan head. The technical settings (gain, contrast, rejection) were optimized after initial evaluation and maintained constant. Real time gated Doppler superimposed on B mode imaging was used for flow measurements. Signals representing artefacts due to erratic movements or forced breathing were discarded and measurements repeated.

Doppler flow velocity tracings representing time-periods of 7s were stored and processed using automated wave form-enveloping duplex.

Flow was studied at the femoral vein 2 cm caudal to the confluence with the deep femoral vein. Peak [PeakV], mean [MeanV] and minimum [MinV] velocities were obtained. Vein diameter was measured by placing the B mode callipers over the proximal and distal intimal-luminal interfaces. Cross-sectional area was calculated from $\pi \times \text{Diameter}^2/4$. Its calculation enabled the estimation of venous volume flow [$Q_{\text{venous}} = \text{MeanV} \times \text{cross-sectional area}$]. Pulsatility index of venous flow [PI_{venous}] was calculated as follows: $\text{PI}_{\text{venous}} = (\text{V}_{\text{peak}} - \text{V}_{\text{min}})/\text{V}_{\text{mean}}$ in keeping with its estimation in arterial flow.¹¹ The velocity amplitude (V_{amp}) was calculated from the difference between PeakV-MinV. At least 4 measurements were performed for each one of the evaluated parameters and then averaged. Venous wall shear stress was calculated from $8 \times \mu \times \text{V}_{\text{mean}}/\text{diameter}$ where blood viscosity (μ) was assumed to be constant at $0.035 \text{ dyn} \cdot \text{s}^{-1} \cdot \text{cm}^{-2}$.¹²

The applied method's reproducibility for the estimated flow in smaller veins such as perforators has been reported ¹³ as well as validation and reproducibility of this automatic method have been described previously.¹⁰

Statistical analysis

Data were expressed as mean and standard deviations and analyzed with the Mann-Whitney-U test for intergroup comparison between obese and non-obese subjects. Correlations between WHR/ waist circumference and venous flow parameters were calculated by Spearman rank test. We did not correlate BMI with flow parameters because BMI was used to differentiate between obese and non-obese subjects and was not a continuous variable since overweight subjects (BMI 25-29 kg/m²) were not included. Statistical analysis was performed using the software SPSS 13. A p-value <0.05 was considered to be significant.

4. Results

All assessed 46 limbs of non-obese (= control) and 44 limbs of obese subjects were included in the analyses. Baseline characteristics are shown in table 1.

Table 1. Baseline characteristics of control and obese subjects

	Controls (n = 23)	Obese (n = 22)	P*
Female	10	10	1.0
Age	49.7 (15.6)	53.5 (15.5)	0.013
Weight (kilograms)	69 (10)	106 (18)	<0.0001
Height (meters)	1.75 (0.08)	1.71 (0.1)	0.2700
Body Mass Index (kg/meter ²)	22.4 (2.1)	36.2 (5.9)	<0.0001
Waist circumference (cm)	88 (10)	122 (11)	<0.0001
Hip circumference (cm)	93 (5)	114 (11)	<0.0001
Waist-to-Hip Ratio (WHR)	0.94 (0.07)	1.08 (0.07)	<0.0001

*All values are mean and standard deviations, * Mann Whitney-U test*

Obesity, hemodynamics and venous diameter

PeakV was significantly higher in non-obese (14.8 ± 7.2 cm/s) compared to obese subjects (10.8 ± 4.8 cm/s, $p = 0.0070$) (Figure 1). MinV tended to be lower in control subjects (-4.0 ± 3.6 cm/s versus -1.7 ± 6.3 cm/sec in obese, $p = 0.056$) (Figure 1). Obesity did not impact MeanV (control subjects 4.8 ± 5.1 versus 3.8 ± 3.5 in obese subjects, $p = 0.239$) (Figure 1). Diameter of the femoral vein was significantly smaller in the control group (7.1 ± 1.6 mm) compared to obese subjects (8.5 ± 2.2 mm, $p = 0.0009$). No significant difference of the flow volume was found between both groups (controls 89.5 ± 81.5 ml/min, obese 99.4 ± 54.7 ml/min, $p = 0.12$). There were no significant differences between left and right lower limb for all assessed and calculated flow parameters (PeakV, MinV, MeanV, diameter, Qvenous, Plvenous, PeakV-MinV) for each group (data not shown).

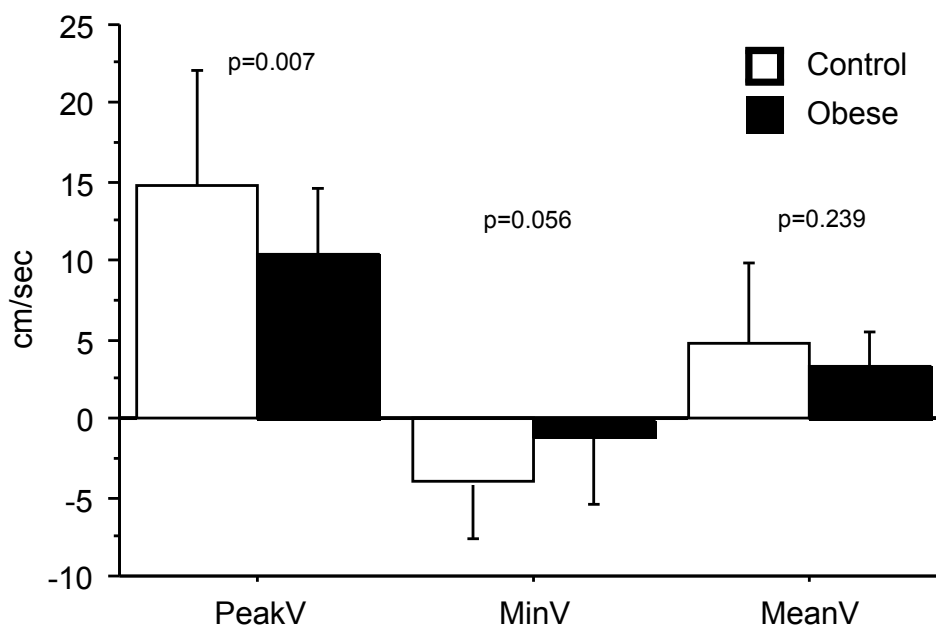


Figure 1

Obesity and outflow resistance / Obesity and shear stress

Pulsatility index indicating outflow resistance was significantly higher in obese subjects (7.24 ± 2.3) than in the control group (5.99 ± 0.86 ; $p = 0.02$) (Figure 2).

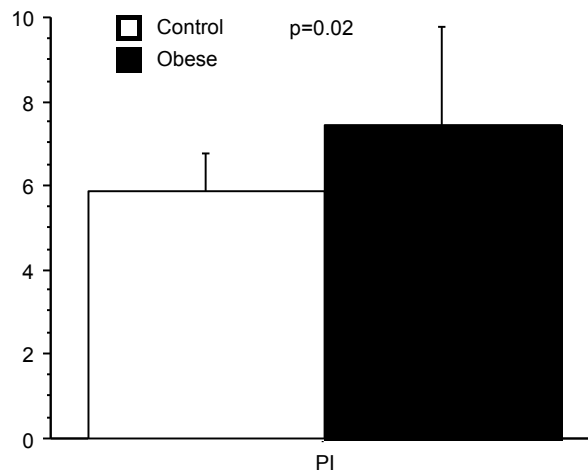


Figure 2

Velocity amplitude (PeakV-MinV) was significantly higher in the control group (18.8 ± 9.4 cm/s) compared to obese subjects (12.5 ± 9.3 cm/s, $p = 0.003$) (Figure 3).

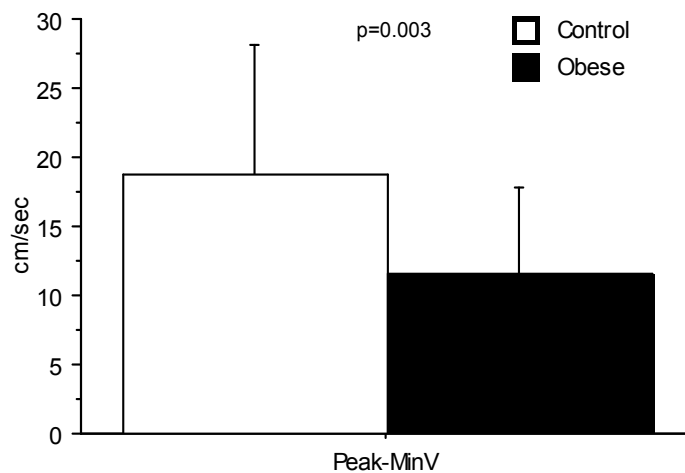


Figure 3

Lower mean velocities and greater diameters result in a lower wall shear stress in obese subjects which was 1.2 ± 0.0 dyn/cm² versus 2.13 ± 2.2 dyn/cm² in non-obese subjects ($p = 0.0172$) (Figure 4).

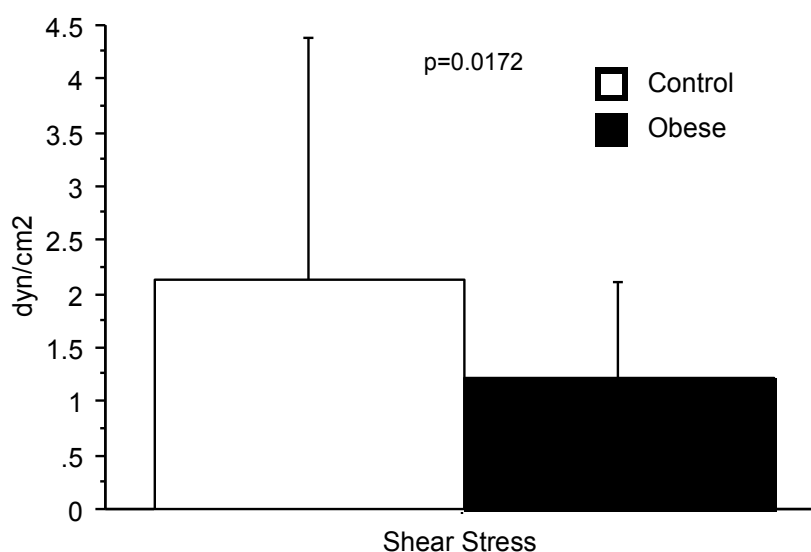


Figure 5

Correlation between abdominal obesity and hemodynamic parameters

Spearman rank correlation revealed a significant inverse correlation between WHR or waist circumference and PeakV, MeanV, velocities amplitude (PeakV-MinV) and shear stress. Furthermore there was a significant positive correlation between WHR/waist circumference for MinV and diameter (Table 2). No significant correlations were found for WHR, pulsatility index and volume flow. However the similar inverse, positive correlations and lack of correlation respectively were found when only waist circumference was taken as an independent factor.

Table 2. WHR and waist circumference in correlation to lower limb venous flow characteristics in 22 obese and 23 non-obese subjects

Parameters	R*	P
Waist-to-Hip ratio (WHR)		
Peak Velocity	-0.44	< 0.0001
Minimum Velocity	0.429	< 0.0001
Mean Velocity	-0.294	0.0084
Peak-Minimum Velocity	-0.533	< 0.0001
Pulsatility Amplitude	-0.197	0.0768
Diameter	0.458	< 0.0001
Volume Flow	0.093	0.4057
Shear Stress	-0.413	0.0002
Waist circumference		
Peak Velocity	-0.378	0.0007
Minimum Velocity	0.314	0.0050
Mean Velocity	-0.25	0.0247
Peak-Minimum Velocity	-0.428	0.0001
Pulsatility Amplitude	-0.153	0.168
Diameter	0.456	<0.0001
Volume Flow	0.143	0.2031
Shear Stress	-0.369	0.0009

* Spearman rank correlation

5. Discussion

The results of the present study suggest that obesity impacts lower limb venous properties. Diameter of the femoral vein was significantly greater in obese compared to non-obese subjects. This could be interpreted as a result of elevated IAP transmitted to the femoral veins and leading to vein wall extension. Increased stasis and reduced forward flow velocity might be a consequence. Indeed, we also found significant different hemodynamic properties between obese and non-obese subjects. PeakV was lower and MinV higher in the obese group most probably due to elevated IAP which results from abdominal fat causing outflow obstruction. Likewise, pulsatility index which is derived from peak, minimum and mean venous flow velocities indicates a higher outflow resistance in obese subjects in our study.

Lower amplitude between peak and minimum venous velocities in obese subjects indicates a continuous flow. This is in agreement with the greater vein diameter found in the obese group and previous reports on intravenous pressure in obese subjects which is greater compared to non-obese subjects.⁷⁻⁹ Abdominal obesity increases IAP which is transmitted to the veins of the lower limbs. This leads to greater tension on the venous vessel wall and hence greater diameters. Minimum venous velocity is usually slightly negative since venous valves close due to backflow. Although one might expect higher venous backflow in obese subjects our data indicates the opposite. It seems more likely that through the greater pressure venous vessel wall is permanently under greater tension in obese people and hence venous elasticity is attenuated. This potentially results in venous valve dysfunction over time and might explain the higher incidence of venous insufficiency in obese people.¹⁴ The significantly lower venous shear stress in the obese group support that increased IAP

in obese subjects results in venous stasis of the lower limb given by correlation between WHR or waist circumference with velocity parameters and diameter. We used BMI for group definition (obese versus non-obese subjects) since this marker of overweight and obesity is established and correlates well with total body fat content in adults.¹⁵ However, this marker fails considering the distribution of fat.¹⁶ In view of the relationship between central obesity and coronary heart disease waist circumference and WHR are accepted. We therefore used these two parameters since this considers abdominal fat distribution.

Our data show a significantly lower value of shear stress at the femoral vein in obese compared to non-obese subjects. Laminar shear stress can promote the release of factors reducing inflammation and the formation of reactive free radicals while low or zero shear stress can promote the opposite.¹⁷ And as furthermore described by Bergan *et al.* decreased shear stress might support development of chronic venous insufficiency.¹⁷ In addition, these alterations of inflammation and coagulation at the venous endothelium are further enhanced by systemic changes in these parameters which in addition to chronic venous insufficiency favour a thromboembolic event.

Whereas there are only few reports on human in-vivo assessment of venous flow properties there are abundant studies showing that obesity increases among other intrinsic factors such as previous VTE, venous insufficiency, chronic heart failure, immobility and cancer the likelihood for an adverse event.¹⁸ Numerous studies have shown a clear relationship between obesity and the risk of idiopathic VTE and pulmonary embolism independent of other recognised risk factors with odds ratios of 2.42 in two separate studies comparing BMI >30 kg/m² with BMI <25 kg/m².^{6,15,19,20}

Even more striking is the fact that waist circumference exceeding 100cm confers up to a 4-fold increased risk of venous thromboembolism.^{19,21} Likewise, obese patients are more likely to develop the post-thrombotic syndrome following VTE.¹ Excess

body weight is not only a risk factor for a first VTE event but also for recurrent VTE events as recently reported.²²

Many researches in this field focused on changes in coagulation and reported that levels of D-dimer, fibrinogen, factor VIII and factor IX are significantly increased according to categories of BMI. Interestingly, stasis as a third factor in the Virchow's Triad has not been as extensively assessed in human in-vivo study. There are invasive human in-vivo studies that investigated the effect of abdominal obesity on IAP and intravenous iliac pressure.^{8,9} These studies required either a venous access or a bladder catheter. In contrast CCDS is non-invasive and allows an assessment of flow patterns. Fronek *et al.* have used CCDS to investigate the effect of aging on lower limb venous hemodynamic.²³ Delis *et al.* used CCDS scan in the assessment of venous hemodynamic of perforator veins as well as to determine the effect of posture on deep vein flow patterns.^{10,13}

Although we studied a rather small group of obese and non-obese subjects the findings are striking considering the limited number of examined lower limbs and underline that abdominal obesity is associated with lower limb outflow impairment. Our study sample is too limited to stratify venous flow impairment for categories of BMI. Furthermore it does not allow drawing any conclusion on venous velocities in patients with moderate overweight (BMI 25-30 kg/m²). Our data only indicates that an impairment of lower limb venous outflow is observed in obese subjects but not whether this might be translated into an increased risk for VTE or CVI. This link has recently been reported by larger, event-driven cohort studies^{18, 19, 21, 22}. However other factors such ambulatory activity, ankle-joint function and gait pattern might play a role as well.

The non-blinded manner of data assessment by duplex sonography must be considered as a shortcoming in our study protocol. Blinding the observer was

impossible in our study setting. Measurements were strictly standardized to overcome it. This standardization and the applied exclusion criteria were also needed to minimize the impact of other factors that might affect venous flow like movements, posture or respiration pattern.

We found no differences between the right and left limb regarding the assessed duplex sonographic hemodynamic direct and indirect parameters (data not shown). It has been shown that iliac vein compression is much more prevalent in women than in men ²⁴. Of course it would have been interesting to provide more precise information about the venous system of the included subjects in our trial, i.e. by means of venous pressure measurements, plethysmography and abdominal CT scanning.

Nevertheless, our data indicates that a simple, non-invasive assessment by means of duplex sonography is sufficient to detect differences in flow patterns with respect to obesity. Although, there was no matching for age between obese and non-obese subjects. We cannot exclude this as a possible confounder even if it does not seem very likely.

In conclusion, our findings support the hypothesis that obesity has a relevant influence on lower limb venous hemodynamic parameters. If this may explain the increased risk for chronic venous insufficiency and venous thromboembolism in obese subjects remains speculative and should be further investigated.

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8. Curriculum vitae

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